therefore considered one of skill in the cytokine arts (*see* paragraph 1 of Dr. Migone's Rule 131 Declaration and the accompanying Exhibit A). Further, Dr. Migone's declaration contends that after reading the specification, she understood that the CRCGCL receptor protein demonstrates homology to the IL-2 receptor common gamma chain, that the CRCGCL receptor protein is only expressed in activated T-cells (*see* Dr. Paul A. Moore's Rule 131 Declaration, discussed below), that the CRCGCL receptor protein possesses a Jak box, and was found to interact with a Jak kinase (*see* paragraphs 5, 6, 8, 11, 12 and 13 of Dr. Migone's Rule 131 Declaration). Dr. Migone also attests that upon consideration of the specification, she understood that the specification asserts that the CRCGCL receptor protein can be used to diagnose or treat immune and autoimmune disorders, specifically, for example, that antagonists to CRCGCL receptor protein can be used to inhibit the proliferation of T-cells (*see* paragraphs 14, 15 and 17 of Dr. Migone's Rule 131 Declaration). This understanding was based upon the teachings of the specification as well as what was known in the field of cytokine research at the time the application was filed (*see* paragraphs 7, 9, 10, 16 of Dr. Migone's Rule 131 Declaration and the accompanying Exhibits B, C, and D).

Therefore, Applicants respectfully submit that the present specification clearly asserts a utility to one of skill in the art that is also specific, substantial and credible.

B. The Examiner also expressed concern over the assertion made in the specification that the claimed CRCGCL receptor protein's expression is "...in only activated T-cells..." (*see* page 8, lines 21-23 of the specification). The Examiner asserted that if Applicants could provide evidence that the expression of the claimed CRCGCL receptor protein was limited to *activated* T-cells, as opposed to resting T-cells, that this would be evidence of a credible assertion of the claimed protein's utility to diagnose or treat immune and autoimmune disorders, specifically, for example, that antagonists to CRCGCL receptor protein can be used to inhibit the proliferation of T-cells. It is well-known in the immunological arts that the activation of lymphocytes generally (which includes B and T-cells) includes both proliferation and differentiation of lymphocytes (*see* Abbas, A. K., et al., Cellular and Molecular Immunology, pages 9-10, 2nd Edition (1991), submitted herewith as Exhibit E).

Applicants submit herewith the executed Rule 131 Declaration of Dr. Paul A. Moore. Dr. Moore asserts that experiments were performed in which the CRCGCL receptor protein's mRNA expression profile in both resting (unactivated) and activated T-cells were isolated and analyzed (*see* paragraphs 6 and 7 and Exhibit A of Dr. Moore's Rule 131 Declaration).

As shown in Exhibit A, the CRCGCL receptor protein is upregulated approximately 10-1000 fold in activated T-cells over resting T-cells. Therefore, Applicants respectfully submit that Dr. Moore's Rule 131 Declaration confirms the assertion made in the specification that the claimed CRCGCL receptor protein's expression is limited to only activated T-cells.

II. Rejections Under 35 U.S.C. §102

Applicants note that in view of the Examiner Interview Summary Sheet for the interview conducted on December 12, 2001, the Examiner has reconsidered and withdrawn the 35 U.S.C. §102 rejections, for which Applicants thank the Examiner.

Conclusion

In view of the foregoing remarks, Applicants believe that this application is now in condition for allowance.

If there are any fees due in connection with the filing of this paper, please charge the fees to our Deposit Account No. 08-3425. If a fee is required for an extension of time under 37 C.F.R. § 1.136 not accounted for above, such an extension is requested and the fee should also be charged to our Deposit Account.

Respectfully submitted,

Dated: February 8, 2002

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